



# **Armed Forces College of Medicine AFCM**



# ***Reabsorption of Bicarbonate & Secretion of Hydrogen***

**by**

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# INTENDED LEARNING OBJECTIVES (ILO)



By the end of this lecture the student will be able to:

1. Define sites of  $\text{HCO}_3$  reabsorption
- 2-Describe mechanism of H secretion
- 3-Explain role of kidney in control of acid – base balance
- 4-Describe how the kidney can regenerate new  $\text{HCO}_3$
- 5- Mention the important buffers in the tubular fluid

# Introduction

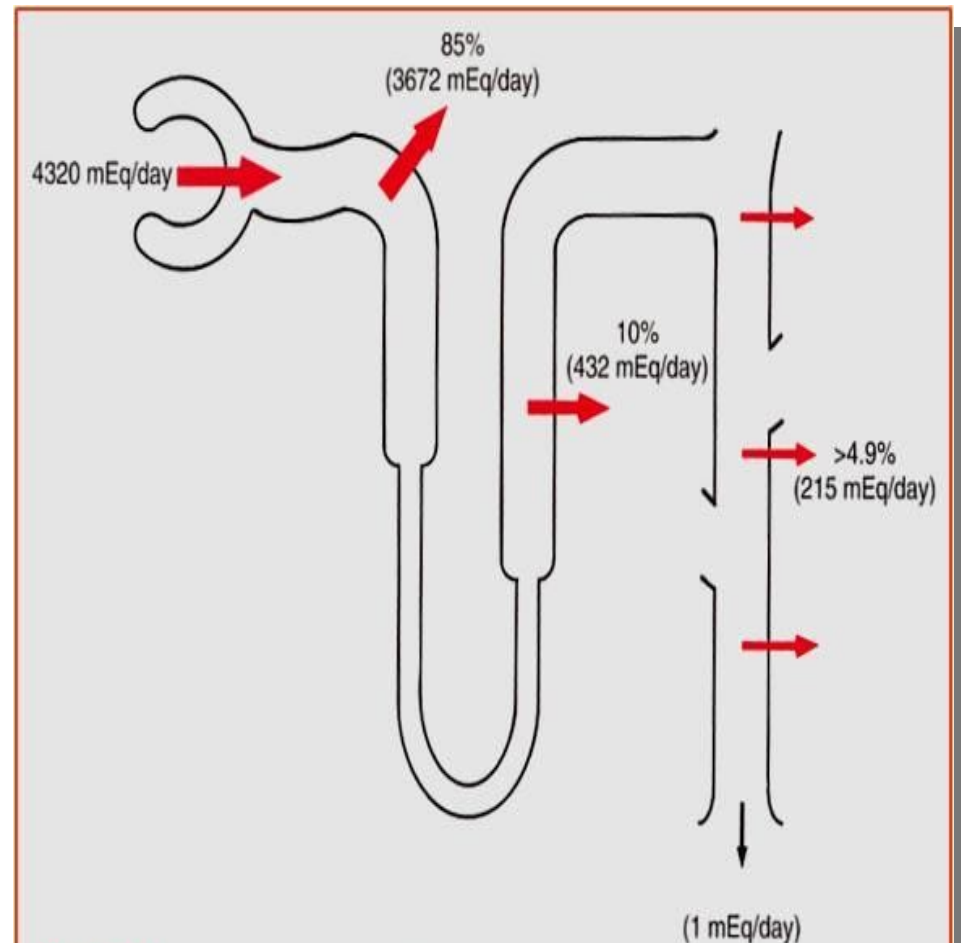


- For each  $H^+$  secreted, one bicarbonate is reabsorbed. Bicarbonate is reabsorbed mainly by the proximal tubule (85%), thick ascending loop of Henle (10%) and collecting duct (4.8%)

# Site of Hydrogen Secretion



- $H^+$  is secreted in all parts of the renal tubule except the descending and ascending thin limbs of the loop of Henle.



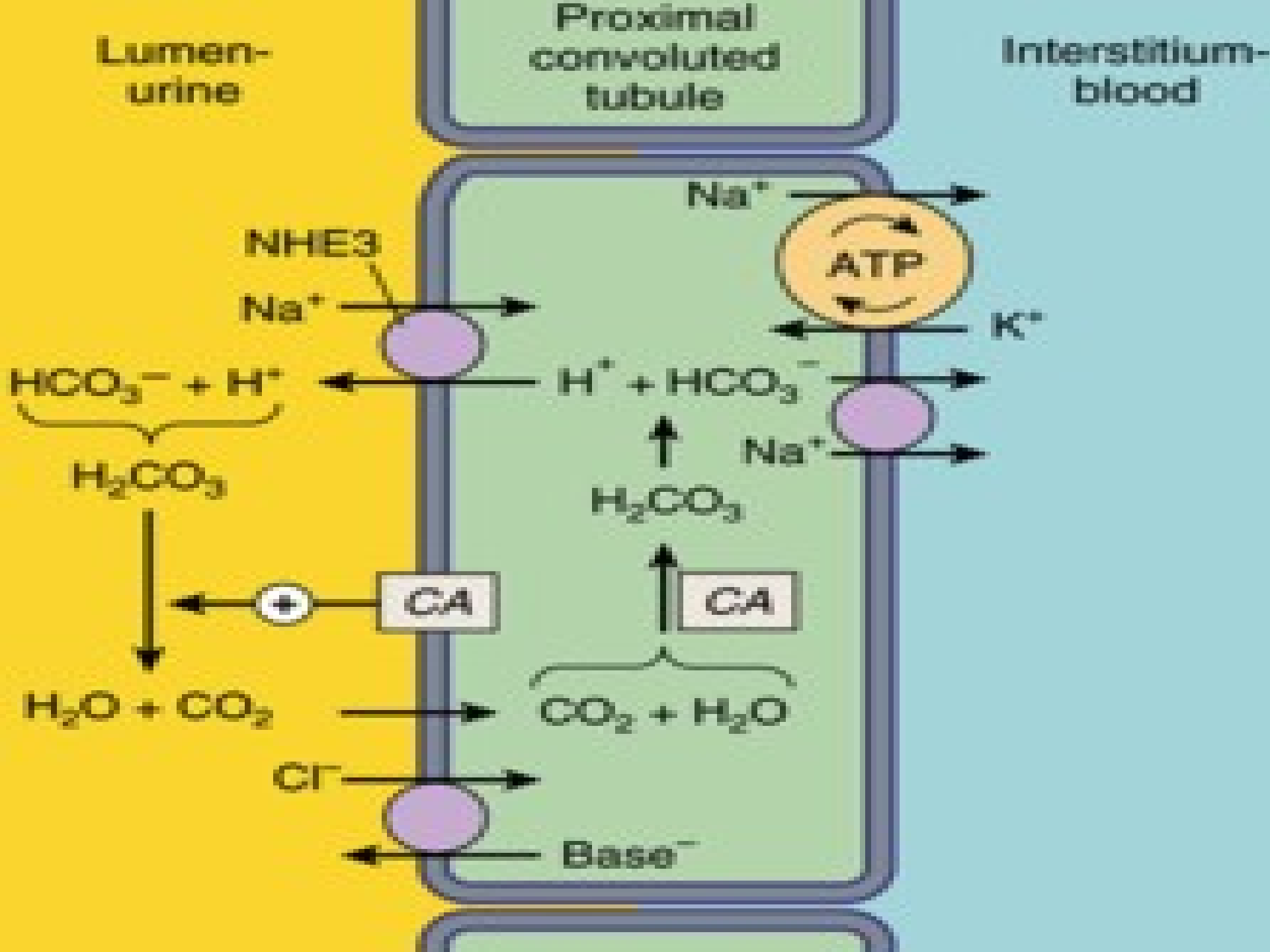
**Figure 30-4.** Reabsorption of bicarbonate in different segments of the renal tubule. The percentages of the filtered load of bicarbonate absorbed by the various tubular segments are shown as well as the number of milliequivalents reabsorbed per day under normal conditions.

# Mechanism of H<sup>+</sup> secretion



## 1. Secondary active transport:

- Occurs in the proximal tubule, thick ascending limb of loop of Henle and initial part of distal tubule.
- The secondary active transport of H<sup>+</sup> occurs by counter-transport mechanism .



# Mechanism of $H^+$ secretion

## 2. Primary active secretion:

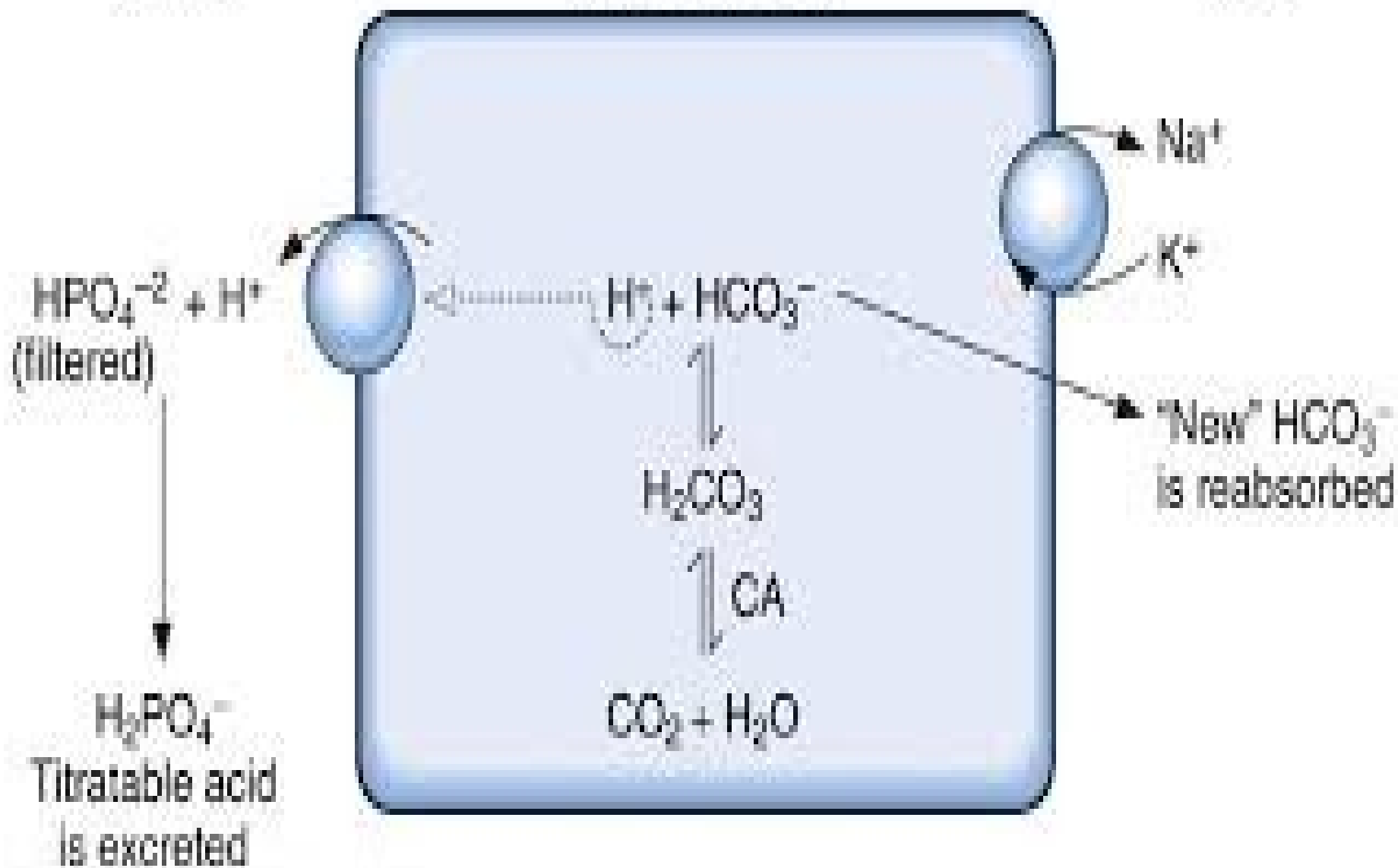
- Occurs in the late distal tubule and collecting ducts by I- cells.
- It is  $Na^+$  - independent.
- It is stimulated by aldosterone, which can be increased up to 900 folds.
- $H^+$  is transported actively by  $H^+$ -ATPase pump at the luminal membrane of the intercalated cells.



Lumen

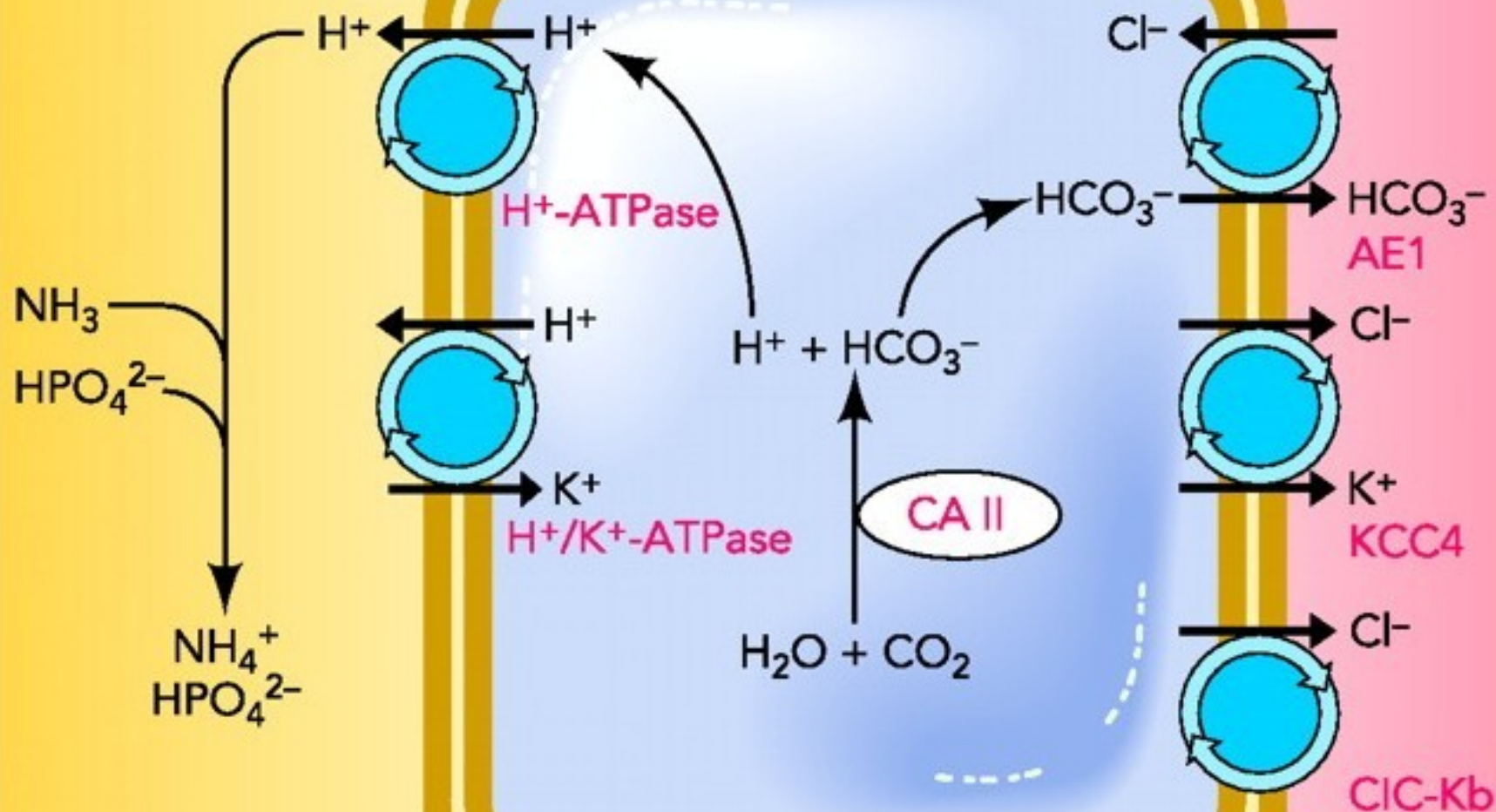
Intercalated cell

Blood



Tubular lumen

Blood



# Fate of $H^+$ secreted



- The secreted  $H^+$  is buffered by the buffers in the tubular fluid.

## **1-In the PCT:**

- Buffering by the  $NaHCO_3$  in the tubular fluid. The net effect is reabsorption of 85% of filtered  $NaHCO_3$ .

# Fate of $H^+$ secreted



## 2. In the Distal tubule and collecting duct:

### a) Buffering by phosphate buffer:

- 30 - 40 mEq of  $Na_2HPO_4$  are available.
- $H^+$  is buffered as follows:
- $H^+ + Na_2HPO_4 \rightleftharpoons NaH_2PO_4 + Na^+$
- $NaH_2PO_4$  is excreted accounting for most of the titratable acid.
- This process results in secretion of  $H^+$  and net reabsorption of newly synthesized  $HCO_3^-$

# Fate of $H^+$ secreted

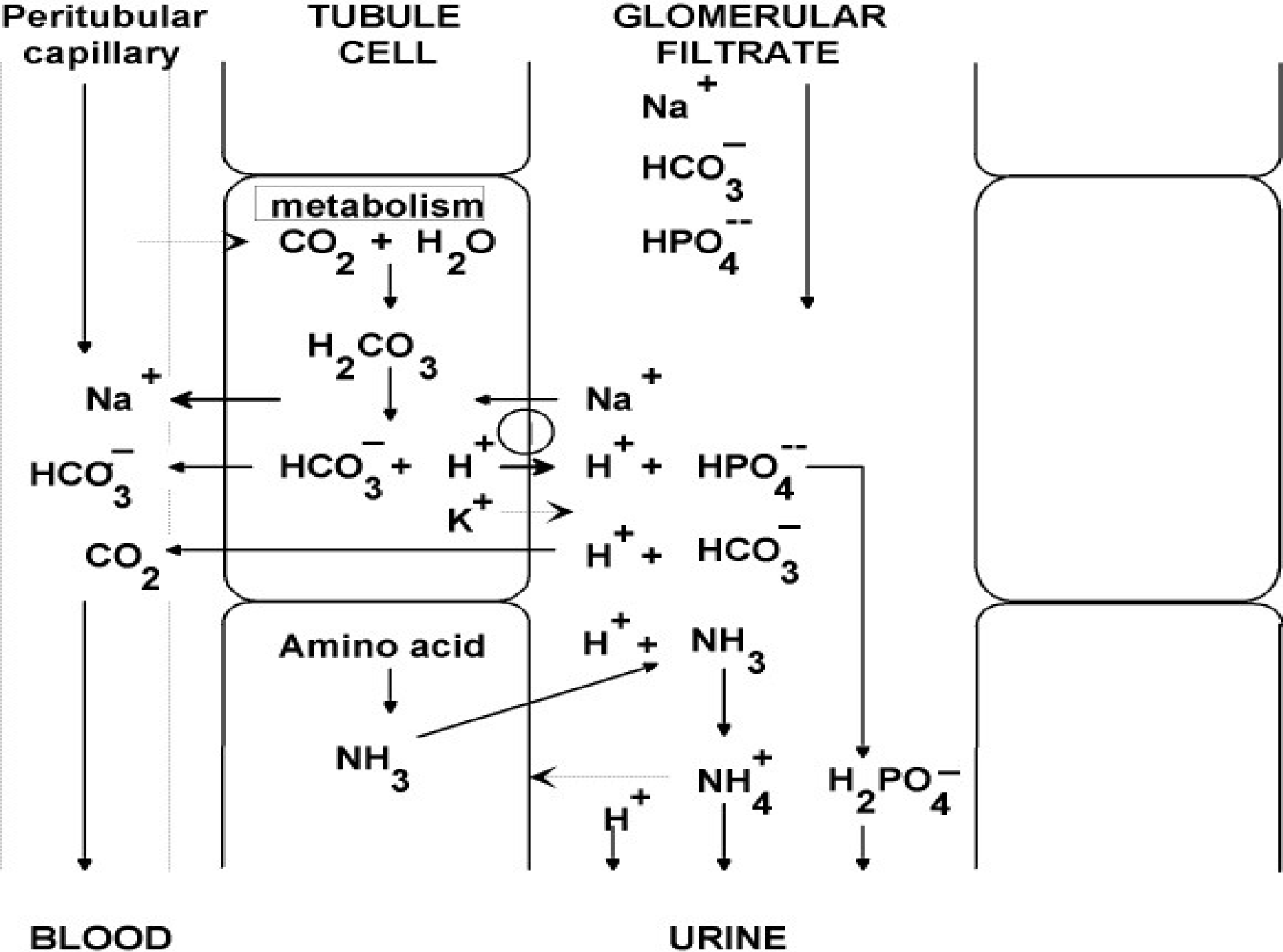


## b) Buffering by ammonia ( $NH_3$ ):

- $NH_3$  is formed in most parts of the renal tubule from glutamine.



- $NH_3$  is lipid-soluble and diffuses into the tubular fluid.  $H^+$  combines with  $NH_3$  to form  $NH_4^+$ , which is then excreted in urine together with  $Cl^-$  (from  $NaCl$ ) forming  $NH_4Cl$ .  $Na^+$  are reabsorbed together with the  $HCO_3^-$  adding new  $NaHCO_3$  into blood.



# Importance of $H^+$ buffering



$H^+$  secretion in the distal tubule and collecting ducts occurs as long as the pH of the fluid in these segments is above 4.5 which is the limiting pH for  $H^+$  secretion. If the secreted  $H^+$  is not buffered, this limiting pH would be reached rapidly leading to stoppage of further  $H^+$  secretion.

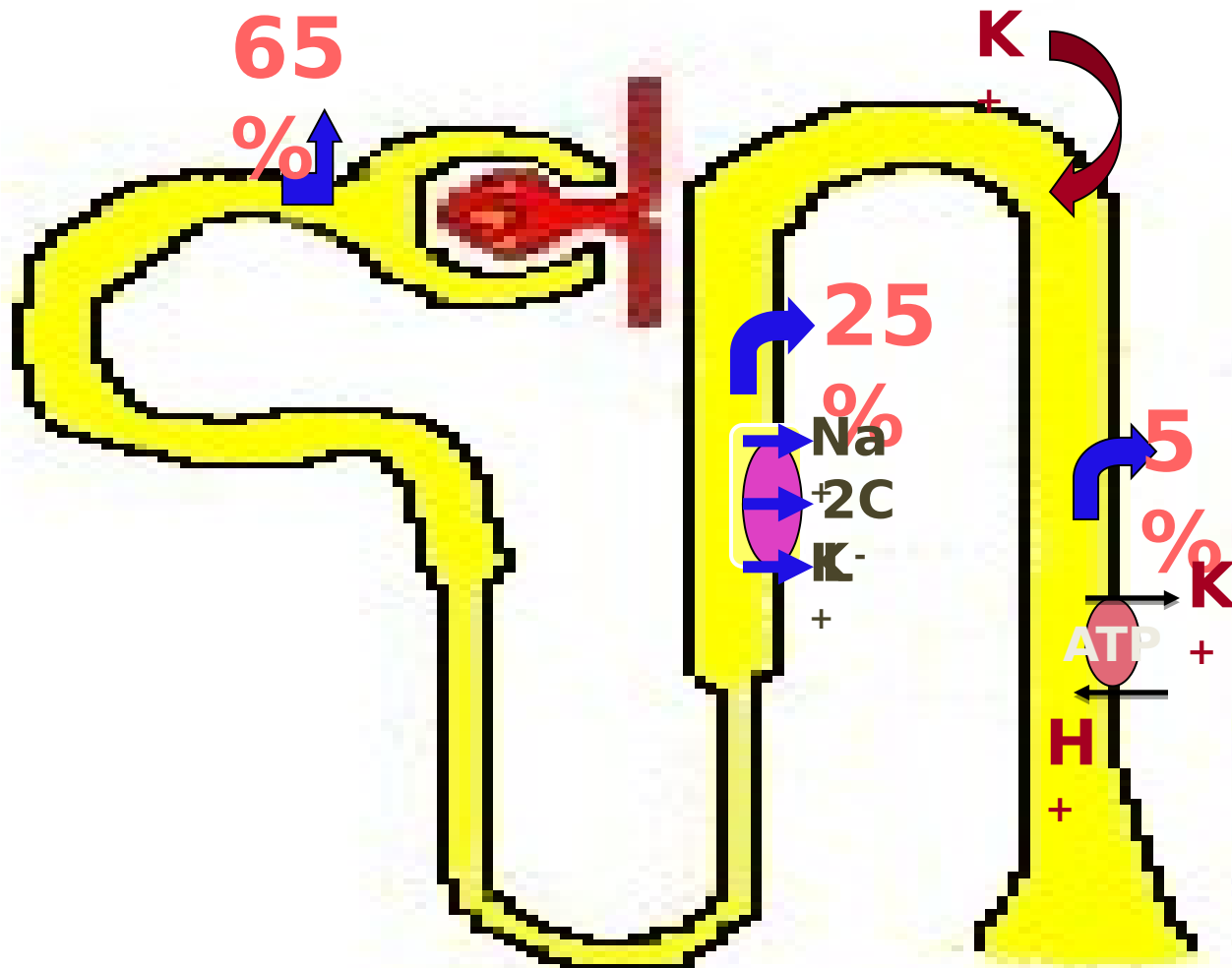
# Factors affecting acid secretion



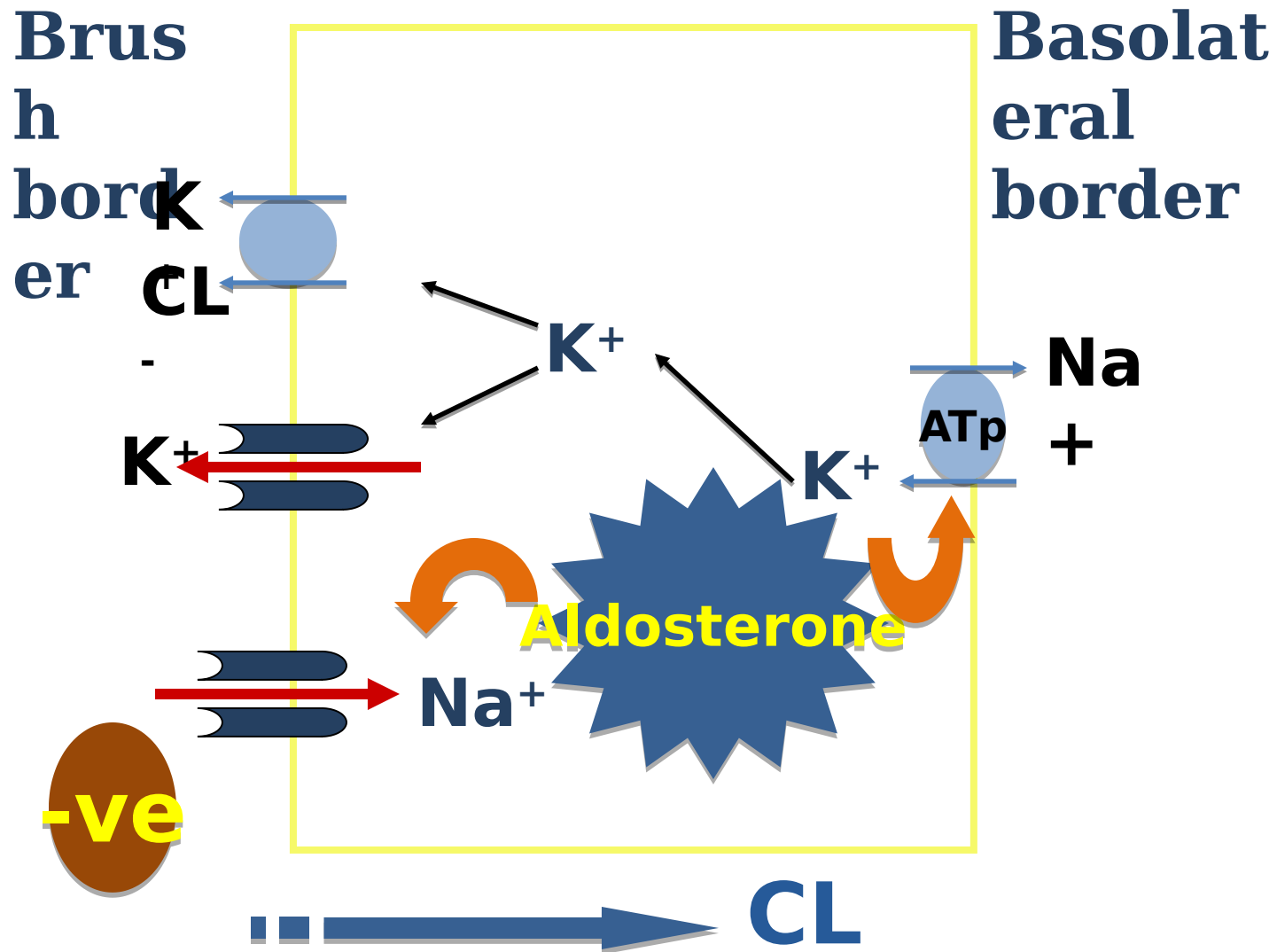
- 1) Aldosterone: increase  $H^+$  and  $K^+$  secretion.
- 2) Intracellular  $PCO_2$ : when  $PCO_2$  is high (respiratory acidosis) more intracellular  $H_2CO_3$  is available and  $H^+$  secretion is enhanced.
- 3)  $K^+$  concentration in the cells:
  - a)  $K^+$  depletion in the cells enhances  $H^+$  secretion.



# K<sup>+</sup> reabsorption



# Potassium secretion by Late DCT & C.



# Regulation of $K^+$ Excretion



1. Plasma  $K^+$  concentration.

↑ Plasma  $K$

2. Flow rate in the distal tubule.

3. Acid-base status. ↑ Activity of  $Na^+-K^+$  ATPase

↑  $K^+$  channels by Aldosterone

Direct effect

Aldosterone

# Metabolic acidosis

